acid,[1-3] and absorption of fermentation products into the blood causes the acidosis.[5] Administering vancomycin hydrochloride or neomycin can normalize the colonic flora, with resolution of the D-lactic acidosis.[1,3,4] Schoorel and coworkers in 1980, instead of using antibiotics, recolonized the fecal flora with gram-negative bacteria with satisfactory results.[2]

Duran and associates in 1979 described a case of D-lactic aciduria in a mentally retarded child who had neither intestinal disease nor had had a bowel operation.[6] A similar case of D-lactic acidosis was recently reported in a 60-year-old man who had not undergone a surgical procedure of the bowel and did not have a history of diarrhea.[7] The source of D-lactic acid in those two cases is speculative.

In the case reported here, there was no evidence of abnormal colonic flora. The treatment consisted of neither dietary change, administration of antibiotics, nor recolonization of the fecal flora. Removing the gastrostomy tube, which had been misplaced in the transverse colon, resolved the D-lactic acidosis and the diarrhea.

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Repeated Pentostatin (2'Deoxycoformycin)-Induced Remissions in a Patient With Advanced Chronic Lymphocytic Leukemia

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BECAUSE OF ITS PREVALENCE and relatively favorable prognosis (median survival of more than five years), chronic lymphocytic leukemia (CLL) is the most common type of leukemia in the western world.[1] In advanced disease, combination chemotherapies including alkylating agents and steroids produce responses in more than 60% of patients.[2-8] More aggressive regimens have not produced superior results and have been associated with increased morbidity.[9,10] Most patients eventually become refractory to standard agents and die of complications related to overwhelming systemic disease. Treating advanced disease is very difficult because of severe anemia, thrombocytopenia, and neutropenia that are often made worse with cytotoxic therapy. There is a need to identify new effective agents for treating this disease. We describe the case of a patient who, despite previous treatments with multiple regimens, had repeated near-complete remissions following brief courses of the intravenous (IV) administration of pentostatin (2'deoxycoformycin).

ABBREVIATIONS USED IN TEXT

ADA = adenosine deaminase
CLL = chronic lymphocytic leukemia
Ig = immunoglobulin
IV = intravenous

Report of a Case

In 1973 at age 59, chronic lymphocytic leukemia was diagnosed in this man. The diagnosis was later confirmed by the typical clinical syndrome and the classic immunologic phenotype with CLL cells expressing immunoglobulin (Ig) M and IgD with only λ light chains and the T65 antigen.[11,12] Between 1973 and 1977 he received an undefined amount of chlorambucil. From 1977 to 1985 he was followed at the San Diego Veterans Administration Medical Center. His disease indices and therapies during that period are shown in Figure 1. In 1979 he had progressive anemia with an increasing lymphocyte count. The monthly administration of chlorambucil was started at single doses of 30 to 40 mg per m^2 and prednisone at 20 mg per day for five days each month. He had a good response but in June 1979 was admitted with respiratory failure and hilar adenopathy. Transbronchial biopsy tissue was histologically consistent with a drug-induced hypersensitivity reaction. A regimen of daily prednisone was started, and his respiratory condition returned to normal. When his disease began to progress, the prednisone therapy was discontinued.

Therapy was restarted with cyclophosphamide, 750 mg per m^2 given IV on day 1; vincristine sulfate, 2 mg given IV on day 1; and prednisone, 80 mg a day by mouth for seven days in three-week cycles. He had a good response, but after six cycles respiratory failure and hilar adenopathy again developed, attributed to a recurrent lung hypersensitivity reaction due to alkylating agents. With the discontinuation of therapy, all symptoms disappeared; the vincristine and prednisone regimen was later discontinued because of disease progression.

By late 1981, he had accumulated a substantial tumor burden. Experimental therapy with a murine monoclonal antibody, T101, was given without a sustained response.[13] He was then given a single agent, bleomycin sulfate, 15 units IV weekly, again with a good response. This, too, had to be discontinued, however, because of respiratory difficulty. After his disease progressed, he had additional trials of T101 monoclonal antibody therapy followed by a regimen of fluorouracil, without response. Splenic irradiation, bleomycin-vincristine, prednisone, and cyclophosphamide were given serially with some decrease in tumor burden, but they also produced severe thrombocytopenia and transfusion-dependent hypoplastic anemia.

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In November 1983 he received the investigational agent, pentostatin (2'deoxycoformycin; Parke-Davis, Division of Warner-Lambert Company, Ann Arbor, Michigan), at a dose of 4 mg per m^2 IV. He received two weekly doses accompanied by an immediate decrease in the lymphocyte count and resolution of his lymphadenopathy. He continued to have anemia with severe neutropenia and thrombocytopenia; a bone marrow examination revealed a severely hypoplastic marrow with a few residual lymphocytes. Two months later, following a pronounced reticulocytosis, his hematocrit and platelet count returned to normal. Despite normal leukocyte and differential cell counts and resolution of all measurable disease, a true complete remission was not obtained based on lymphocyte immunologic phenotyping that showed persistence of the malignant clone.[14] His leukocyte count remained in the normal range for six months. When his tumor burden began to increase, a single 4-mg-per-m^2 dose of pentostatin was given in October 1984. There was an immediate decrease in all measurable tumor, and the leukocyte count remained in the normal range for five months.

No further treatment was given until August 1985. At that time a single infusion of pentostatin decreased the leukocytes from 113,000 per μl to 3,800 per μl in less than a week. As with previous treatments, there was no toxic reaction except for mild nausea and vomiting and some malaise. This time his leukocyte count remained in the normal range for two months. He was retreated in October 1985 with a single injection while his leukocyte count was still in the normal range, but the differential count showed persistent lymphocytosis.

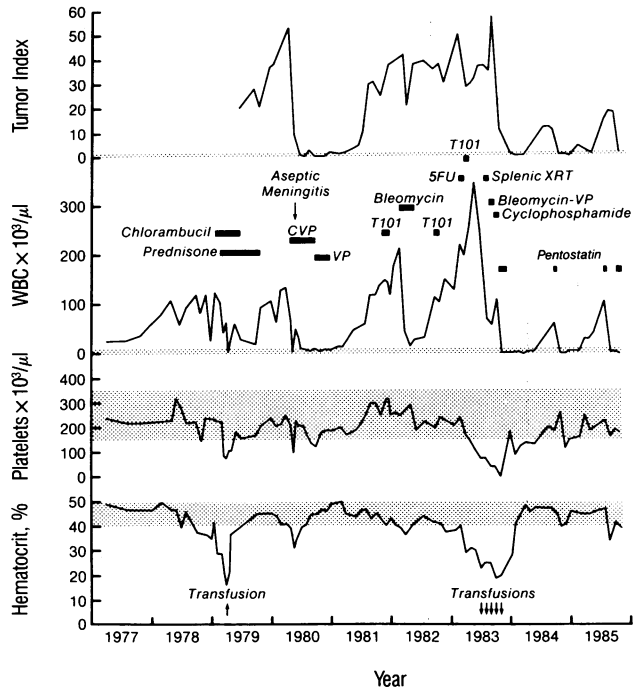

Figure 1.—The graphs depict the clinical course of this patient with chronic lymphocytic leukemia. The tumor index was calculated by adding the sum of the products of cross-sectional diameters of the largest lymph nodes in bilateral cervical, axillary, and inguinal regions, plus the number of centimeters below the left costal margin of the spleen. The shaded areas represent the normal range for each measurement. CVP = cyclophosphamide, vincristine sulfate, and prednisone, 5FU = fluorouracil, T101 = T101 murine monoclonal antibody, VP = vincristine sulfate and prednisone, WBC = leukocyte count, XRT = radiation therapy

Shortly thereafter, while in clinical remission, the patient was admitted to hospital for a severe autoimmune hemolytic anemia that required intensive care. Corticosteroid therapy was started. *Staphylococcus aureus* bacteremia developed, and the patient responded with a granulocyte count of more than 20,000 per μl. Subsequently he had an apparent drug-induced agranulocytosis that resolved within four days of discontinuing cefazolin therapy. By then, however, he had gram-negative sepsis and respiratory failure and died after six weeks in hospital. A bone marrow study done during his neutropenia showed erythroid hyperplasia but no evidence of chronic lymphocytic leukemia other than one lymphoid aggregate. There was no recurrence of lymphocytosis or lymphadenopathy during the hospital stay. Thus, he died while in a clinical remission of his leukemia, although the immune hemolytic anemia, which precipitated a series of complications leading to his demise, was probably due to the persistent underlying immunologic defects associated with CLL.

Discussion

Many studies have verified the correlation between clinical stage in CLL and survival,[15-17] although some authors have suggested that patients who respond to chemotherapy do live longer.[18] This case is noteworthy because of the dramatic remissions that were obtained with the use of pentostatin despite previous chemotherapy with six different agents, splenic irradiation, and experimental immunotherapy. With only sporadic retreatment, this patient maintained an excellent performance status for two years after being treated at a far advanced stage of disease. The quality of this remission and the limited therapy required to achieve it deserve special emphasis. This patient, who was transfusion-dependent and had severe thrombocytopenia, apparently on the basis of marrow replacement rather than an immune basis, regained completely normal blood values after receiving pentostatin. Whereas previous therapies had resulted in several admissions for marrow suppression and infections, once this man had achieved his initial remission, repeated therapy was not required and he had no secondary complications from the chemotherapy.

Pentostatin is a nucleoside analogue produced by *Streptomyces antibioticus.*[19] It is a potent inhibitor of adenosine deaminase (ADA), an enzyme in the purine salvage pathway responsible for deamination of adenosine to inosine and deoxyadenosine to deoxyinosine. The observation that children with congenital ADA deficiency had lymphopenia and immunodeficiency led to the suggestion that ADA might be required for lymphocyte proliferation. Thus, it was postulated that an inhibitor of ADA such as pentostatin might be useful in the treatment of lymphoproliferative disorders. In vitro, in the presence of deoxyadenosine, pentostatin induces cytotoxicity associated with increased cellular levels of deoxyadenosine triphosphate[20,21] that leads to decreased DNA synthesis by feedback inhibition of ribonucleotide reductase.[22,23] Pentostatin, however, is also toxic to nonreplicating cells[24,25]; thus, the mechanism of toxicity is not entirely due to deoxyadenosine triphosphate effects on DNA synthesis.[23] Pentostatin plus deoxyadenosine induces DNA strand breaks in nondividing lymphocytes,[26] but the mechanism of DNA break is unknown. Although pentostatin is generally more toxic to T lymphocytes, which have higher levels of ADA than B lymphocytes, it is toxic to CLL cells as well.[27,28] Other mechanisms of cytotoxicity are probably in-

volved, in that CLL cells have lower ADA activity than normal lymphocytes,[29] but adenosine and its analogues are more toxic to those cells than to normal lymphocytes.[30]

Although initial pentostatin trials from England were unassociated with toxicity, further studies have shown substantial side effects.[31-38] Phase I studies in the United States in children with leukemia and adults with lymphoproliferative disorders and other malignant diseases at doses of 0.25 to 1.0 mg per kg per day for three to five days were associated with nephrotoxicity; pulmonary edema; hepatic toxicity; and severe central nervous system toxicity, including lethargy, confusion, seizures, and coma.[31-34] These reactions were generally reversible when the drug therapy was discontinued. Lower doses, such as 4 mg per m^2 given IV for three days in cases of cutaneous T-cell lymphoma and 4 mg per m^2 given IV weekly in patients with CLL have been much better tolerated.[33,35,38] CLL cells have much lower levels of ADA; thus, smaller doses of pentostatin can produce a clinical effect. Grever and co-workers reported one complete response in seven previously treated patients and no significant toxicity.[37] They recently reported on treatment of 28 patients with CLL, with five objective responses and no significant toxicity.[39]

As shown by these other studies, not all patients with chronic lymphocytic leukemia can be expected to have a dramatic response to the administration of pentostatin. Phenotypically, this patient had the classic form of the leukemia, with cells that expressed IgM and IgD λ immunoglobulin on their surfaces as well as the T65 antigen.[11,12] This phenotype was unchanged throughout his treatment course, and, even at times of clinical complete remission, circulating surface immunoglobulin-positive/T65-antigen-positive cells could be detected in the circulation.

Another interesting historical feature of this patient is that he had responded to various approaches with chemotherapy and had not clearly become resistant to these agents. Rather, their use had to be discontinued because of severe pulmonary hypersensitivity reactions. Thus, he may have been potentially more responsive to pentostatin than a patient who had become refractory to standard chemotherapy agents. In the other single-institution trials of the use of pentostatin in CLL, most patients have been heavily pretreated or refractory to prior chemotherapy.[33] In view of the response of this patient, the issue of enhanced responsiveness in previously untreated patients might be raised.

Another unique feature of this patient was that he had received several treatments with the murine monoclonal antibody T101. While this antibody has not produced significant clinical remissions in patients with CLL,[13,40] it is conceivable that treatment with this agent sensitized cells in some manner so that they were more susceptible to pentostatin. This is particularly intriguing in view of the activity of pentostatin and CLL in T-cell diseases, and the fact that the T65 antigen is specific for T cells but it also reacts with B-cell CLL.[11]

Finally, in vitro this patient's cells were exquisitely sensitive to pentostatin, with a 50% lytic concentration of 0.27 μmol per liter on day 4. His cells had relatively high levels of adenosine deaminase activity at around 6 μmol per liter per minute per 10^{10} cells.[20] It may be that CLL cells from different persons are heterogeneous for susceptibility to ADA inhibition by pentostatin. If additional investigation supports this association, there may be an in vitro assay predictive of a chemotherapy response that would be more easily reproduced and more reliable than the disappointing clonogenic assay.[41] In fact, a regimen of fluorouracil was tried in this patient because of promising results in a CLL clonogenic assay (courtesy of Raymond Taetle, MD), but there was no response clinically.

The dramatic responses achieved in this patient with minimal treatment were particularly gratifying in contrast to the usual experience in such patients who have been heavily pretreated and who have anemia and thrombocytopenia. Additional studies in untreated and minimally treated patients with CLL seem appropriate on the basis of this experience and that in chronic lymphocytic leukemia, hairy cell leukemia, and other B-cell malignant diseases.[27,39,42,43] Furthermore, additional studies may be warranted in previously heavily treated patients if accompanying in vitro studies can be done to confirm or refute the association of a clinical response with in vitro cytotoxicity.

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An AIDS Diagnosis Used as Focus of Malingering

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PSYCHIATRIC DISORDERS are often associated with medical problems or are offered by a patient or physician as an explanation of "medical" symptom complexes.[1,2] A medical evaluation usually and appropriately ensues to explain a patient's symptoms, but a serious consideration of psychiatric diagnoses is often delayed.[3,5] The acquired immunodeficiency syndrome (AIDS) is a new syndrome caused by the human immunodeficiency virus (HIV) and marked by uncommon infections, neoplasms, and other still poorly defined disorders. We present a patient with AIDS who indicated he had symptoms attributable to an organic disease but who ultimately was diagnosed as having a psychiatric disorder in which he was using his AIDS diagnosis to meet other psychosocial needs.

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ABBREVIATIONS USED IN TEXT

AIDS = acquired immunodeficiency syndrome
CT = computed tomographic
HIV = human immunodeficiency virus
VAMC = Veterans Administration Medical Center

Report of a Case

A 29-year-old homosexual man was admitted to the Denver Veterans Administration Medical Center (VAMC) with new, progressive, left-sided weakness. The patient reported that he had recently moved from Texas where a diagnosis of AIDS had been made. He said he had had numerous opportunistic infections including *Cryptococcus, Pneumocystis,* and cryptosporidiosis. He had received several experimental drugs, including suramin sodium and difluoromethylornithine (DFMO). He brought numerous records with laboratory and diagnostic test results that were positive only for an elevated cytomegalovirus titer. There was no record of an HIV titer.

He reported having increasing left hand weakness about two weeks before admission. Six days before admission, he experienced a stabbing, bilateral temporal headache. He presented to the Denver VAMC six days later. The left-sided weakness had gradually increased to the point of difficulty in holding objects. The headache was accompanied by a brief episode of loss of consciousness. This was followed by nausea, dizziness, confusion and disorientation, and left arm and leg paralysis. The patient also described transient diplopia and a central scotoma.

The patient said he did not have auras, apraxias, incontinence, tonic-clonic movements, drug use, a history of a similar episode, or a history of diabetes mellitus. He had had numerous electroencephalograms in the recent past that were reportedly normal. He had no known allergies and was currently taking no medication. He had attempted suicide in 1984 with an amitriptyline overdose. He had hepatitis in 1976 and genital herpes of an uncertain duration. The patient was an unemployed chauffeur who lost his job after being diagnosed with AIDS. He had a 15-year history of promiscuous homosexual activity. He smoked cigarettes and used alcohol and marijuana occasionally. He said he did not use drugs intravenously.

He was muscular and appeared healthy. There was no fever, his blood pressure was 110/70 mm of mercury, respiratory rate 12 per minute, heart rate 90, weight 93 kg (205 lb), and height 190 cm (6'3"). He had a gold circular earring in his left nipple. The neurologic findings varied with the observer. Cranial nerves I through XII were completely intact except for a weak right sternocleidomastoid muscle. There was a false-positive orbicularis oculi test. Motor strength on the right was normal, but all muscle groups tested in the left upper and lower extremity were weak (0 to 4/5). He was able to stand and walk on both heels and toes. Muscle tone was notably increased in all groups tested with active resistance to movement, relaxed with persistence, and then was normal. A sensory examination showed no abnormalities. Deep tendon reflexes were normal. Cerebellar function was normal except for the left extremities, which could not be tested. He had a leukocyte count of 5,200 per μl with a normal differential, and the sedimentation rate was 11 mm per hour. The platelet count, prothrombin time, and other routine laboratory tests were normal.